Neonatal Isoerythrolysis

A PowerPage Presented By

Neonatal isoerythrolysis (NI) occurs relatively frequently in neonatal foals and is a common source of questions on board exams. NI is characterized by destruction of a foal's red blood cells (RBC) by alloantibodies of maternal origin, absorbed via colostrum. The goal of this PowerPage is to review the pathophysiology, clinical signs, diagnosis, treatment, and prognosis for NI.

Key Points

- Most often affects foals born to multiparous mares
- Occurs in foals less than 7 days of age and typically cases present at 2-3 days of age
- Mucous membranes may be tinged yellow and the sclera may appear icteric
- The most common erythrocyte antigens implicated in NI are the **Aa** and **Qa** factors

Pathophysiology of Neonatal Isoerythrolysis

- NI is an immunologic type II hypersensitivity reaction between RBC antigens on the neonatal foal's RBCs and antigen-specific antibodies, produced by the mare, that are ingested by the foal when consuming colostrum (for more information on hypersensitivity reactions, see the *Hypersensitivity Reactions PowerLinks).*
- The most common erythrocyte antigens implicated in NI are the **Aa** and **Qa** factors.
    - These "foreign" RBC antigens that the fetus expresses are inherited from the sire and NOT possessed by the mare
- In order for a mare to produce antibodies against RBC antigens, she must first be exposed. This exposure to foreign RBC antigens (that is RBC antigens that she does not express on her own RBCs) may occur from prior blood transfusions, exposure to fetal blood from placental abnormalities early in gestation or from exposure to fetal blood during parturition.
- Donkeys have a unique "donkey factor" RBC antigen not expressed by horses; thus, mule foals have a higher incidence of NI.
- In summary, the three things that must be present for a foal to develop NI are:
    - Neonatal RBC antigen inherited from the sire that the mare does not express
    - Mare does not express neonatal RBC antigen but has developed maternal antibodies to that specific neonatal RBC antigen
    - Neonatal ingestion of colostrum

Clinical Signs

Signs are typically seen in neonatal (< 7 days of age) foals and reflect poor oxygen content of the blood:

- Tachycardia
- Tachypnea, dyspnea, lethargy
- Icterus
- Development and severity depends on amount of antibodies absorbed

Left: 2 day old foal with icteric sclera and mucous membranes

Diagnosis

- Based on signalment and clinical signs
- Supportive clinicopathologic information includes hyperbilirubinemia and anemia
- Confirmed by cross-match of the foal's RBCs and the mare's serum
  - "Jaundice foal agglutination test" (observing if agglutination occurs between foal RBCs and mare's colostrum)

Treatment

If NI is recognized when the foal is less than 24 hours old:

- Withhold the dam's colostrum (i.e. muzzle the foal)
- Provide an alternate source of milk and passive transfer

If greater than 24 hours of age:

- Monitor the foal's packed cell volume (PCV) and the rate of decline
  - As a general rule, if the PCV falls below 12%, a blood transfusion is indicated
  - If the PCV is low (i.e. 18%) but stable, the clinician may opt for conservative monitoring. If the PCV does not decline any further over the next 24 hours, a blood transfusion may not be necessary
  - If the PCV continues to decline, preparation for blood transfusion should be made
  - Good supportive care is also indicated. Stress and exercise should be minimized

Prognosis

- NI is a treatable disease with most foals recovering with proper treatment and blood transfusions.
- If severe anemia (i.e. < 10%) is present when the clinician evaluates the foal with NI, the time necessary to acquire blood for a blood transfusion may be limited and may contribute to mortality.
- Client education of future pregnancies is necessary to prevent the disease from occurring again.